to offer. I confess I have none. I have tried many, many things, internally and externally; all sorts of cough mixtures; all sorts of sprays and inhalations, including the globe inhaler, with vaporized oils and divers drugs, and finally, I have adopted what precedes, as best. Of course, judicious tonic medication, with iron, bark, cod-liver oil, arsenic, strychnine, etc., are at times desirable, and undoubtedly indicated. But above all, let it be understood that nothing practically equals dry or moist vapor inhalations of creosote, after the manner I have so frequently affirmed. Indeed, for almost all affections of the respiratory tract, including laryngeal and pulmonary tuberculosis, there is absolutely nothing now known quite or at all equal to them in efficacy and great power for good. But in order to get good, the best, results, one must have practical knowledge how to use them, and faith in their power to help when other things without them fail or are incomplete.

In the way of spa treatment, the only resorts I believe in specially on the eastern side of the United States are Sharon and Richfield Springs, giving the preference to the former, despite the fact that it is not an amusing place. Aix-les-Bains and Homburg, abroad, are the two valuable springs in my judgment. At Aix, one gets Marlios vaporizations; at Homburg, a specially useful innocuous alkaline spring, the Elizabethan, to drink.

AN ANATOMICAL STUDY OF PERICARDITIS.[1]

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Pericarditis is among the most venerable of recognized cardiac lesions in the history of medicine. Galen is said to have observed pericarditis in animals, and to have suspected its existence in man. Among the ancients the "hairy heart" was supposed, according to Haller, to indicate great valor and bravery. Among those heroes who after death were found to be so endowed are said to have been

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[1] Read at a meeting of the Pathological Section of the Buffalo Academy of Medicine, April 20, 1909.

Leonidas and Lysander. Morgagni and Vieussens described the obliterative type of pericarditis, and Corvisart, in 1811, noted the significance of the bulging pericardium. Although Laennec was familiar with the lesion, he expressed his doubts as to the possibility of diagnosis, and the characteristic to-and-fro rub of the acute condition was not recognized until 1824 by Collis.

Notwithstanding the antiquity of the anatomical recognition of pericarditis, Osler confesses that it is a condition more frequently recognized in the dead-house than in the ward, referring chiefly to chronic pericarditis. Great difficulty in diagnosis of the acute lesion should no longer exist, unless it occur as a terminal condition in the late stages of some disease when the clinician's attention for detail oftentimes becomes relaxed in the study of obviously fatal cases. In such instances, however, the recognition or its failure is of little practical importance.

During the past three years our attention has been especially drawn to the subject, for the most part, because we have found the lesion so frequent at the autopsy table, and so rarely recognized by us in the wards. Yet the conditions in which pericarditis arises as a frequent complication are in general well understood and fully recognized. We have been constantly on the watch for it in such instances, but nevertheless our percentage of ward diagnoses in cases subsequently demonstrated in the dead-house has been disappointingly small. This study has, therefore, been undertaken not only in order that we might, if possible, correct these errors of diagnosis in the future, but also especially that we might more satisfactorily determine the nature of the process and better estimate the bearing of so marked and striking a lesion, which, however, often exists with very few or no obvious clinical signs.

The cases on which this study is based have been for the greater part taken from our own personal service; a few are from the routine protocols of the pathological department at the University and Bellevue Hospital Medical College. Our clinical observations have been chiefly made in the service of one of us at the City Hospital. In all, 1000 protocols have been examined, and of these, 150 have shown pericarditis.

Only frank and outspoken pericardial lesions of true inflammatory nature have been considered in this study. In the acute cases, for example, no instances of hydropericardium have been included, and we wish to make a sharp distinction between pericarditis, a true inflammatory lesion, and the hydropericardium, which occurs in various œdematous conditions. This may, indeed, be followed by true inflammation, but it is not in itself primarily of this nature. Of the chronic, only such instances have been included as showed manifest inflammatory thickening of the membrane or adhesions of considerable extent. The small granulomatous patches which are found so frequently, especially over the auricles and cardiac base, for example, have not been included in these statistics, although these changes are also, in our opinion, unquestionably inflammatory.

CLASSIFICATION AND PATHOLOGICAL ANATOMY. For clinical purposes pericarditis is best classified as acute, subacute, and chronic. Subacute or chronic pericarditis occurs in many instances as an extension or terminal state of the acute forms in which there has been a fibrous or granulomatous replacement of the acute exudate.

In both acute and chronic forms the process is usually found most marked on the visceral layer of the membrane, even in those cases in which the lesion has been produced by direct extension, as from a pleural, pulmonary, or mediastinal inflammation. The most logical explanation of this fact appears to be the greater physical activity and the greater vascularity of the epicardium, as compared to the parietal layer of the membrane.

The acute forms are subclassified as fibrinous, serofibrinous, hemorrhagic, and purulent. We have found the division into fibrinous and serofibrinous unsatisfactory, since in both instances the lesions are practically identical. In the one, more clear serum is exuded; in the other, this serum is richer in coagulated fibrin; we shall, therefore, in this study group together all cases ordinarily classed separately. Similarly, hemorrhagic pericarditis is almost without exception the simple adding of extravasated blood cells to the serofibrinous exudate or to a purulent one, and we have, therefore, for statistical purposes considered hemorrhagic cases under the heading of one or the other of these forms. By some authors; however, hemorrhagic pericarditis has been considered as diagnostically definite; thus, Thorel states that it is present in tuberculous tumors of the pericardium, in uremia, the various hemorrhagic diatheses, and in leukemia. We have found it to have even a broader etiological basis and to be shown in practically any severe type of pericardial inflammation. Stewart,[2] for example, reports it present in a case of infection of the pericardium by the colon bacillus.

Acute serofibrinous pericarditis is a lesion characterized by the primary appearance of minute granulomas on the surface of the pericardium. The capillaries are injected and proliferation of the endothelial and connective-tissue cells is set up, together with inflammatory infiltration of the entire depth of the membrane. The change is seen most frequently or most marked over the base of the heart and notably over the walls of the auricles. Only the epicardial layer may be primarily involved, but sooner or later a similar change appears in the adjacent parts of the parietal layer. At the outset the lesion is very apt to possess a patchy character, but eventually in progressive instances it finally becomes diffuse. Coincident with the inflammatory changes mentioned a deposit, first, as a thin pellicle of fibrin gathers over the eroded surfaces, and this may increase in thickness, varying in different instances from a very delicate web to a coat as much as 1 cm. in thickness. Serum and leukocytes exude into the sac from the distended capillaries, and the amount which may so collect may become very great, as much as 2 to 3 liters, according to Osler and Gosselin, while Kestner claims to have found from 3 to 10 liters in scorbutic cases—all amounts far in excess of the fluid which can be artificially injected into the normal sac post-mortem, as shown by the experiments of Smith, Lusk, and others. Minute petechial hemorrhages appear in a good many instances, either into the fibrinous deposit or more frequently into the subendothelial connective-tissue layer, and in some cases actual granulation tissue may appear. Of course, when the pericarditis develops in such conditions as the hemorrhagic diathesis, in purpura or scurvy, the hemorrhagic exudate is very pronounced. Hemorrhage is also frequent in tuberculous inflammation and in those inflammations which arise from neoplastic invasion of the pericardium. In these last-mentioned circumstances blood extravasation takes place directly into the free fluid, and is not limited to the substance of the membrane or its adherent exudate.

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[2] Zentralbl. f. innere Med., 1904, xxv, 863.

It is generally admitted that in the healing of serofibrinous pericarditis fibrous substitution of the exudate takes place, and when abraded surfaces are apposed, adhesion, of course, with resulting synechia at these points follows.

The bacteria found in serofibrinous pericarditis commonly bear a direct etiological relation to the process. Those most commonly present are the streptococcus, the pneumococcus, various staphylococci, and, according to Poynton and Paine, Diplococcus rheumaticus. Secondary infecting organisms of all sorts may, of course, be present, and in some cases the exudate is sterile.

Purulent pericarditis in our experience occurs either from direct purulent infection of the pericardial sac or as a sequence to the serofibrinous form when infection with pyogenic organisms takes place. In most instances the effusion is less abundant than in the cases of serofibrinous inflammation, but it may become great before death ensues. As a rule, especially when the condition follows the serofibrinous form, the exudate is rich in fibrin, although the shreds are apt to become more or less liquefied. Pus, eroded connective tissue and endothelial cells, and general inflammatory detritus are also abundantly present in the fluid. The portions of the membrane previously most deeply covered by fibrin are now commonly overlaid by granulations. The color of the purulent effusion is, of course, chiefly dependent on the nature of the infecting organisms. Gas may also appear due to the action of microbic growth. Whenever the case has been of long standing one is very apt, in addition to whatever organisms may have been first present to find the colon bacillus or members of the proteus group. We have twice found Bacillus aërogenes capsulatus in purulent pericardial effusion when general infection with this organism was present; gas was, of course, abundant in both these cases. The tubercle bacillus, streptococci, and staphylococci are the most common organisms found, and they usually bear a direct etiological relation to the disease.

When healing follows purulent pericarditis, extensive fibrous adhesions almost invariably form, and in some cases we have found patches of calcification.

Chronic pericarditis is most conveniently divided into a chronic fibrous variety, in which adhesions either do not exist or are present to very limited extent, and those cases which show chronic fibrosis with adhesions. The latter is by far the most frequent form in our experience. In the simple chronic fibrous form the essential process is a thickening of the fibrous layers of the membrane due in many instances to inflammatory changes, but frequently to a general fibrosis with this local manifestation, as in certain types of syphilis, akromegalia, arteriocapillary fibrosis, and the like. Hess[3] thus describes this lesion as a local manifestation of a general chronic serositis. The thickening may be entirely limited to a single layer of the pericardium, in which instance it is almost invariably the visceral layer which is so involved, but in most examples, and especially when it follows a previous serofibrinous or purulent inflammation both layers are diseased, but the visceral to the greater degree. Occasionally one finds extensive plaques of thickening without general involvement of the sheet, and here the changes are most prominent about the base and over the auricles.

Microscopically, the alterations are briefly those of a fibrous hyperplasia, usually with more or less still active round-cell infiltration, and commonly with exudation of some extent. In this variety, and also in cases of fibrous pericarditis with adhesions, when the etiology is tuberculosis or syphilis, the characteristic lesions of these infections may be present, although by no means invariably.

In chronic pericarditis, with adhesions, a very wide range exists between those cases in which only a few fibrous strands unite the visceral and parietal membrane and those in which the union is complete and of dense scar tissue. In our statistical studies we have placed in this class only those cases which showed fairly extensive synechia, but it is, of course, manifest that all degrees of adhesion may exist, depending probably on the nature and extent of the primary process and on its rapidity of absorption or replacement. In our experience when the union has been incomplete we have found the most numerous adhesions immediately about the apex or just over the auricles, but in this particular the cases vary greatly.

Tuberculous Pericarditis. As has been stated by Osler, pericarditis caused by tuberculosis may manifest itself either with distinct and demonstrably tuberculous lesions, as miliary tubercles or caseous masses, or it may be found as a simple fibrous thickening of the membrane with or without adhesions. Although in the latter case the change is unquestionably indirectly due to the tubercle bacillus, if the specific bacilli or distinctly tuberculous changes cannot be demonstrated, we have chosen to class such examples as chronic fibrous or adhesive pericarditis, due to tuberculosis and not as true tuberculous pericarditis. In most instances of this nature it has been found that the pericardial change is but a local example of a more or less general fibrosis. As will be shown later in our study, tuberculous infection of the pericardium may cause practically any sort of anatomical pericarditis, from serofibrinous or hemorrhagic to purulent, fibrous, or adhesive, a fact which we think should be more generally recognized. This may depend on the varying degree of virulence of the organism, on the resistance of the body and its reaction, but probably still more on the nature of the contaminating organisms.

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[3] Eine Klinische-experimentelle Studie, Marburg, 1902.

In tuberculous pericarditis of the chronic variety the membrane is, in our experience, generally much thickened and is studded with yellow necrotic areas, which are softening tubercles. When, on the other hand, the change is but a chronic stage of an acute tuberculosis of the serosa, as in some slow cases of general miliary tuberculosis, obliteration of the cavity may not be present, but only occasional shreds of adhesion are demonstrable. In practically all cases of tuberculosis of the pericardium, tubercle formation is not diffusely present, but is associated with inflammatory changes of clearly simple nature, and organisms other than the tubercle bacillus are also etiologically concerned. Indeed, in most cases, from a study of the lesions one is impressed with the idea that the tubercle bacillus is here a relatively inactive organism, and that other agents are probably much more directly involved in the process.

Syphilitic Pericarditis. Practically the same conditions obtain as in tuberculosis. Unless distinctly syphilitic lesions can be demonstrated in the pericardium, an admittedly rare condition, the lesion should not be classed as syphilitic pericarditis, although, as we show later on, various forms of pericarditis may be caused by syphilitic infection, a fact not generally recognized. There is now no doubt that any of the various anatomical forms of pericarditis, either acute or chronic, may be caused by the syphilitic virus. As might be expected, however, only a relatively small proportion show typical syphilitic pictures. In these, accompanying syphilitic changes in the myocardium are the rule. In passing, however, it is interesting to note that in the two instances of gumma of the heart, which occurred in the service of one of us, no pericardial changes of note were present. In the average case of syphilitic pericarditis the membrane shows a more or less diffuse thickening, and it is only on microscopic examination that changes suggestive of syphilis appear. It is manifested usually by perivascular infiltration, nuclear fragmentation, and a distinct tendency to necrosis, as well as toward hyperplasia of the connective tissue. It is probably partly for this reason that syphilitic pericarditis has been so rarely reported. Gumma of the pericardium has never occurred in our experience, and McPhedran states that but three cases have been recorded.

Traumatic pericarditis does not deserve a separate classification from the pathological standpoint, since in most instances the actual pericarditis is due not to the traumatism, but to infecting agents introduced with it. It occurs mostly in surgical practice, and the changes induced depend on the nature of the contributory infection. The cases of perforation of the pericardial sac with foreign bodies from the œsophagus and stomach are very interesting, and the consequent pericarditis is invariably of a purulent type. The elder Flint thus cites in his text-book a case following perforation and infection from the swallowing of false teeth. Personally, we have never seen a case of this nature in man, but it is common in the ruminants, and several years ago one of us reported such a case in a buffalo, when the perforating body was a bale wire, quite a common circumstance, I am told, among domestic animals.

Mechanical pericarditis also does not exist as a distinct pathological entity, although it is a clinical condition of some importance. It is seen most frequently as a serofibrinous inflammation, occurring in such conditions as dilatation of the heart or from overaction, oftentimes with more or less dilatation. It is seen, for example, in Graves' disease, two marked instances of which have been observed in our service. Although the most common anatomical change found in these cases is a serofibrinous inflammation, fibrous hyperplasia also occurs with relative frequency. Rubino produced acute pericarditis in animals by experimental trauma, and we all have noted its frequency in tube-fed animals.

Knox[4] describes a further form of pericarditis characterized by the appearance of small fibrous nodules along the course of the arteries of the epicardial layer, not entirely unlike the syphilitic form described by Balzer.[5] According to Knox this pericardial inflammation is caused primarily by lesions of a degenerative character in the walls of the coronary arteries. We have not personally observed this form, except, perhaps, in those cases due to marked coronary arteriosclerosis, when the superjacent epicardium was thickened, hyperplastic, and more or less infiltrated; we have not considered such instances as frank pericarditis in this study.

ETIOLOGY. It is very questionable if a primary idiopathic pericarditis exists. At least, if it ever occurs it is a very rare condition, and even in reported instances Thorel[6] discusses the probability of most of the recorded cases being tuberculous or the result of hemic infections of other varieties. It is certain then that pericarditis is, almost without exception, a secondary lesion or a complication in some other disease or dyscrasia. This statement is now generally accepted, and holds true except for the relatively unimportant traumatic cases. In general our study upholds the usual conceptions in regard to the etiological factors chiefly concerned in the production of pericarditis, but in the investigation of the literature of the subject we have been impressed with the fact that in most reports the statistical conclusions are based on clinical findings only, or in some instances on bacteriological investigation alone, and are, therefore, less apt to be accurately based than when the data have been derived from complete postmortem examinations aided in many cases by histories, clinical notes, and bacterial examinations, as in our statistics.

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[4] Jour. Exp. Med., 1899, iv, 245.

[5] Archiv der Phys., 1883, vi, 93.

[6] Ergeb. der allg. Path. u. Path. Anat., 1909, S. 921.

In the acute serofibrinous type of pericarditis lobar pneumonia leads as the most frequent cause, and was present in 26 out of the total of 67 instances. This is in direct contradiction to the statement of von Schrötter, who states that pneumonia is not shown in his observation as an especially frequent cause of pericarditis. This apparent discrepancy is largely explained by the clinical observation, which I am sure has been apparent to all of us, namely, that certain epidemics of pneumonia have been characterized by the frequency of pericarditis as a complication, and in other years other complications have been more common. For example, nearly all the hospital physicians in New York City remarked on the great frequency of pericarditis in the pneumonia epidemic of 1906, during which time my statistics were partly collected.

In most instances pericardial infection in lobar pneumonia has taken place by transmission by direct contact in so far as could be determined by the local condition of the adjacent pleura and mediastinal tissues, although, of course, lymphatic or hemic transmission cannot be excluded in this general infection. Four cases of pericarditis of the serofibrinous type were found to have developed in the course of bronchopneumonia, and three from simple pleurisy without pulmonary involvement.

Rheumatism has been quite generally reported as the most frequent condition in which pericarditis of the serofibrinous variety develops. When we recall that under the head of rheumatism must be included many presumably septic conditions incorrectly diagnosticated, it is readily understood why this disease has been so generally accepted as the most frequent cause of pericarditis. In our observations, which it must be remembered are founded on postmortem and not on clinical evidence alone, rheumatism was found to be the factor in but 7 out of the 67 instances. Von Schrötter reports acute rheumatism as the cause in 30 per cent. of cases, von Harrass in 7 per cent., Pribram in 5.2 per cent., and Eichorst in 3 per cent. We must, however, not fail to recollect that in this respect clinical data may sometimes be the more valuable, since the larger percentage of these instances of rheumatic pericarditis recover, leaving no traces which can be afterward definitely ascribed to rheumatism anatomically. Again, in regard to rheumatism all clinical observers are united in admitting that the percentage of pericarditis as a complication varies greatly in different years. Poynton, in his book, states that he has isolated Diplococcus rheumaticus from the pericardial effusion in a high percentage of cases of pericarditis, and he, therefore, assumes that all these instances are examples of true rheumatic infection. Until the relationship between acute rheumatic fever and the diplococcus of Poynton and Paine has been more definitely demonstrated most of us will be inclined to accept this evidence as far from positive.

Six of our cases developed in the course of general sepsis and two took place in generalized tuberculosis, without, however, the presence of tubercle bacilli or tubercles in the pericardium; in other words, they were the result of the mixed infection.

But five instances of serofibrinous pericarditis developed in the course of renal disease, a considerably smaller percentage than is shown in most statistics, and probably accounted for by the fact that our data were collected from postmortem and not clinical records. Four were due to acute and one to chronic nephritis.

Five cases developed as a result of syphilitic infection, four in the tertiary stage. In these examples no typically syphilitic lesions were demonstrable in the pericardium, although the predisposing influence was clearly syphilitic infection. Alcoholism, acute myocarditis, and asthenia appear as the etiological or determining factors in two instances each. In but one case could no probable etiology be found, but this instance was not submitted to bacteriological investigation.

Finally, in regard to the etiological factors concerned in the production of acute serofibrinous pericarditis, it will be observed that general bacterial infections predominated in a high percentage (46 out of 67).

In our eighteen instances of purulent pericarditis generally septic conditions naturally lead, although this was demonstrable in but seven. Lobar pneumonia with apparently direct transmission of the infection was present in three instances and purulent pleurisy in two. Three formed the terminal picture, following rupture of a tuberculous abscess of the lung with direct invasion of the pericardium, but without the formation of tubercles, although doubtless these would have developed had the patient not promptly died from the resulting general septic condition, of which the pericarditis was evidently but a part. Three cases of apparently idiopathic purulent pericarditis appear in which, while a generally septic condition was present, no other presumably primary focus of suppuration could be discovered.

But eight instances of chronic fibroid pericarditis independent of adhesions occur in our series. Of these, tuberculosis (without tuberculous lesions in the pericardium) leads in four examples, and chronic nephritis appears as the probable exciting cause in two.

In chronic adhesive pericarditis, sixty-one instances of which occur in our list, tuberculosis, which was demonstrable in 17 cases, appears as the most constant etiological factor. In none of these were tuberculous lesions present in the pericardium, and the change seems then to have been induced by the general toxemia of the disease rather than by the immediate action of the tubercle bacillus. This statement is in general accord with the conclusions of most of those who have made a special study of pericarditis in its relationship to tuberculosis. In most instances the fibrosis does not appear to have been the result of a direct transmission of the inflammatory process from the lung and pluera, but to have followed the toxemia of the disease. Osler, for example, states that as a cause of pericarditis tuberculosis follows hard upon acute rheumatic fever.

Arteriocapillary fibrosis, with the pericardial lesion as a local manifestation, appears as the second most frequent cause of this form of pericarditis. It was present in 11 of our cases. We believe that the importance of this etiological factor has been very commonly overlooked by most writers, who have in many instances attributed the changes to nephritic disease, the "uremic pericarditis" of Banti, whereas the renal changes were but a part of the end result of the primary arteriocapillary disease. Independent and clearly etiologically important, nephritis could be traced in but 2 of our cases, while Bulil finds it causative in 35 per cent. of his, Mamberger in 14 per cent., and Frerichs found it probably causative in 18 out of 292. Banti,[7] in two carefully observed cases, came to the conclusion that the nephritic type of pericarditis is caused by the toxic factors present in nephritis, and states that he was able to reproduce the lesion experimentally. It seems highly probable that the frequency of terminal infection in uremia may bear some relationship to the development of pericarditis in nephritic conditions; although perhaps the distant cause of the lesion, it would not be so considered in our study.

Syphilis, with its general tendency to fibrosis and inflammatory changes, has been found as the most probable factor in 10 out of our 61 cases. In none of these, however, could definitely syphilitic alterations be demonstrated in the pericardium, and we, therefore, assume that the pericardial lesions were but local evidences of the general tendencies shown in this disease. We believe that the relationship of syphilis in this respect has been very generally unrecognized by previous writers on this subject, most of whom exclude syphilis as the probable cause unless definitely syphilitic changes are demonstrable in the membrane. This is by no means in accord with the accepted bearing of syphilis on such conditions as hepatitis, nephritis, and myocarditis, all lesions very similar in nature histologically to chronic adhesive pericarditis. Alcholism, with an anatomical picture somewhat similar to the changes found in cases of syphilitic nature, appears in nine instances out of the sixty-one, and we find in going over the literature that the relationship of this chronic drug poisoning has been also very generally overlooked by most authors, many of whom do not even mention it. Even a slight knowledge of the nature of the general changes which occur in chronic alcoholism is such as fully to convince one of its probable important bearing on pericarditis, particularly of this form.

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[7] Ueber urämische Pericarditis, Zentralbl. f. path Anat., 1894, 461.

Chronic endocarditis was concerned in but four examples of pericardial adhesions, in all of which the mechanical factors resulting from irregular action and cardiac dilatation appear to have been prominent.

The relatively small occurrence of rheumatism as an etiological factor in these chronic cases is notable in our series. It was found in only four cases. This probably is misleading, for again our statistics have been mostly founded on anatomical studies, and in some instances the histories were either entirely wanting or deficient in regard to this important question. Further, other chronic conditions are very apt to be associated with rheumatism to such a degree as to distract one's attention from the rheumatic factor; thus, alcoholism, tuberculosis, syphilis, and arteriocapillary fibrosis are all conditions frequently following or associated with rheumatism, any of which present more characteristic and definite anatomical changes. Personally, we are fully convinced that the general opinion in regard to the close relationship especially between rheumatism and the chronic adhesive form of pericarditis is correct.

But 6 cases of true tuberculous pericarditis were found in our series of 150. None of these were primary, and, indeed, primary tuberculosis of the pericardium is a most rare condition in the literature; that is, cases in which actual tubercle bacilli or tubercles have been demonstrable in this membrane and not elsewhere. All of our six instances developed in the course of a more or less generalized tuberculosis, that is, in tuberculous bacteremia rather than in merely localized tuberculosis of the lung or pleura. This fact is especially interesting when we recollect that up to recently it was rather generally conceded that a high percentage of cases of pericarditis with adhesions were really tuberculous. This false impression seems to have originated from the great frequency of tuberculous toxemia as a cause of chronic pericarditis, and because of inadequate anatomical study of the thickened membrane in cases of pericarditis with adhesions. Considering the great frequency of tuberculosis in its relationship to pericarditis, the relative immunity which the pericardium enjoys against this type of inflammation is, indeed, striking.

A very similar condition of affairs exists in regard to syphilis in its relationship to pericarditis, and the relative immunity here, as in tuberculosis, can perhaps be best explained by the theory of McPhedran, who asusmes that it is due to the relatively slight vascularity of the membrane. In but a single case of our 150 could definite acute syphilitic changes be shown. This was an example of early but very severe secondary syphilis, in which death followed from perforation of an acute syphilitic ulcer of the aorta, but in which an acute syphilitic granulomatous pericarditis with inflammation of nearly all the serous membranes was demonstrable. We have already discussed, however, the important relationship which syphilis probably bears to the development of the chronic adhesive form of pericarditis. Rosenthal,[8] however, states that although it is generally assumed that syphilitic pericarditis occurs only in conjunction with syphilitic disease of the myocardium, it may do so independently, and Schönemann also reports such an instance. The cases recorded by Virchow, Ricord, Mracek, and Herxheimer all showed definitely syphilitic involvement of the myocardium.

No discussion of the occurrence of pericarditis in diseases of infancy, as in measles, scarlatina, and the like, has been presented, as our experience has been limited to adults.

Although there is more or less general unanimity of opinion in regard to the conditions in which pericarditis is prone to arise, the immediate local determining factors which in any given case cause the onset of the pericarditis are by no means clearly shown. It has already been pointed out that on account of the relatively small blood supply of the membrane it is rather less likely to become involved in bacteremic processes than other similar membranes, as those of the brain, for example. In this regard the observations of Charrin, however, tend to demonstrate that the metabolic products of bacterial growth when in the proximity of the pericardium tend to favor involvement of the membrane.

The predisposing influence of local traumatism, even though of slight degree, has been apparently fully demonstrated, the principle involved being apparently that of the locus minoris resistentiæ. This well-recognized fact has been corroborated by the experiments of Banti, who first caused irritation of the membrane by the injection of turpentine or by the mere puncture with a platinum needle, subsequently producing an experimental bacteremia with the pneumococcus, when a pneumococcic pericarditis was quite constantly produced. Rubino caused a typical pericarditis by the preliminary infliction of a blow over the pericardium or even over the thorax at large, and the subsequent injection into the blood stream of virulent staphylococci. Somewhat similar experiments have been conducted with the diplococcus rheumaticus by Poynton and Paine. Stern[9] thinks that other and less definite injuries to the thorax may be the determining factor, especially when mediastinal hemorrhages are induced. These observations are in a way corroborated by certain experiments of our own, in which we have noted the great frequency of pericarditis, especially of the serofibrinous form, in animals tube-fed for considerable periods, in which traumatisms to the mediastinal tissues resulted from the frequent passage. Pericarditis is very prone to develop in these animals even when no bacteremia preëxists, although in most of our experiments the general resistance of the tissues was already reduced by drug administration and terminal infection was, of course, thereby favored.

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[8] Berl. klin. Woch., 1900, Nos. 47, 48.

[9] Ueber traumatische Enstehung innere Krankheiten, Jena, 1900.

Clinical observation has also led us to consider the role which overaction of the heart plays, as in Graves' disease, in nephritis with elevated blood pressure, and in the numerous conditions in which rapid action of the heart oftentimes with more or less dilatation of its chambers occurs. Although as yet our data do not warrant us in more than a very tentative statement, we believe that these factors do play a very important role in the determination of pericarditis in cases in which it might not otherwise develop.

THE ASSOCIATION OF OTHER LESIONS WITH PERICARDITIS. In this relationship more interest is derived from the bearing of the concomitant lesions upon the pericarditis than of the pericarditis upon them, for while it may be caused by or follow in the wake of several other lesions, there are very few lesions which follow or result primarily from pericarditis. Nevertheless the association of concomitant lesions in the heart and other viscera with pericarditis becomes at times of diagnostic and prognostic value, more so, however, as indicative of the general process or condition in which the pericarditis has originated than of any direct value in the immediate diagnosis or management of the pericardial change itself.

Entirely as one would expect, the lesions most constantly associated with pericarditis are changes in the pleura. In our 150 cases pleural lesions were present in 136. In well over half of these cases it appears that the pleural change is the more anterior, and that the pericardial alteration occurred later, perhaps even as a result of the pleural lesion, though still back of this relationship is the general disease or condition in the course of which the two membranous inflammations developed.

A direct relationship between pericardial and pleural lesions is best shown in the acute serofibrinous cases, in which out of the total of 67 instances, 60 showed pleural changes; in 36 of these the lesions were identical in character. In 11 others chronic inflammatory changes were present in the pleural sheet, which, nevertheless, also appears to have had a direct bearing on the onset of the adjacent pericardial inflammation. Four cases of purulent pleurisy appear to have been the point of origin of the acute serofibrinous pericarditis, so that in 51 out of 67 cases the pericarditis seems to have appeared as secondary to a like or similar process in the pleura.

This pleuropericardial relationship is even more clearly shown in the cases of purulent pericarditis when associated changes were present in all except one. Purulent pleurisy was present in 9 out of the

18 cases, and in 5 others the pleural change was serofibrinous in nature; possibly, however, in these last five, secondary to the pericarditis. In 3 out of the 4 remaining cases the pleural changes were chronic inflammatory.

The association between chronic adhesive pericarditis and pleurisy is less definite, but the pleura was found involved in 48 out of the 61 cases. In 31 of these the pleural change was like that in the pericardium, which would seem to suggest a common origin. It is very important to note in this regard that in but two instances was the pleural inflammation demonstrably tuberculous when the pericardial change was simply chronic inflammatory in nature. In three examples the pericardial lesion certainly was not secondary, but perhaps rather causative of the pleural change, since the pericarditis was a chronic adhesive one and the pleurisy acute. In six instances both acute and chronic alterations were demonstrable in the pleura.

In our 6 cases of tuberculous pericarditis, tuberculous pleurisy was present in 4, and the other 2 were examples of general miliary tuberculosis, in which for some reason the pleural membranes appear to have escaped invasion.

Although some authors have assumed a more or less direct relationship between pericarditis and endocardial disease, this does not seem to be borne out by the study of our series. From our records it seems even highly problematical if there be any significant relationship between these processes. Thus, of our serofibrinous cases, 46 of which were caused by bacteremia of some variety, only 10 showed acute endocardial changes; 7 of these were unquestionably rheumatic, and bore no other relation to the pericarditis than that both had a probably common etiology. The fact that 28 out of these 67 instances showed chronic endocardial changes is believed to be of no bearing, but to be explained by the great relative frequency of chronic endocarditis. All these cases were very carefully gone into under the supposition that deficient muscular action, with perhaps dilatation and mechanical friction might have predisposed toward the pericarditis. This probability could not be sustained in any of these instances except when other changes to be mentioned later on were present.

This lack of correlation between endocardial and pericardial disease is further emphasized in the purulent cases, which although, universally due to bacteremic conditions were unassociated with endocardial lesions of an acute character, except in one instance. From these cases one may apparently more or less definitely assume that infection is rarely borne by the route of the cardiac vessels from the endocardium to the pericardium, or vice versa.

Acute endocardial alterations were entirely absent in the 61 cases of chronic adhesive pericarditis, but in 35 chronic endocardial inflammation was present, but in no such degree or form in any as to indicate probable dependent or etiological relationship.

Myocardial alterations are present in the majority of cases of pericarditis, but that this lesion has more than an accidental or common etiological relation in most instances does not seem likely. Nineteen of the acute serofibrinous cases were accompanied by acute parenchymatous degeneration of the heart muscle. Study of these shows that in but a very small number was the pericardial lesion in any likelihood responsible for the myocardial disease, which in most cases was clearly due to the general toxemia of the disease in which the pericarditis had developed. On the other hand, a certain number of cases are present in which the dilatation and deficient muscular action of the myocardium consequent upon the myocardial disease may have had a predisposing or determining effect in the evolution of the pericarditis. This suggestion is given further probability by the occurrence of eighteen examples of fatty degeneration of the myocardium in which no derivative effect of the pericarditis can be assumed, but rather the contrary, and this, in our belief, is one of the factors which favors the appearance of terminal pericarditis in the numerous conditions in which myocardial degeneration or deficiency is shown. That fatty degeneration bears an especially important relationship is also suggested by the fact that but six instances of fibroid myocarditis were associated with the pericarditis, a very sharp contrast to the occurrence of the fatty degeneration, which in most cases was further suggestive by the concomitant occurrence of more or less dilatation. In 20 of the acute serofibrinous cases absolutely no myocardial lesions could be made out, and it is most striking to note that only 2 of the 67 showed acute inflammation of the myocardium.

This lack of effect on the heart muscle of the adjacent inflammatory process is still more vividly illustrated in the purulent cases, of which only two showed acute inflammatory lesions in the muscle. Nine of the 18 naturally showed parenchymatous degeneration of greater or less degree, and in 6 fatty degeneration was associated.

All of the 8 cases of chronic fibroid pericarditis without adhesions showed myocardial lesions, among which there were 2 of fatty degeneration, 3 of fibrosis, 2 of brown atrophy, and 1 of an acute and incidental parenchymatous degeneration.

Of the 61 cases of chronic pericarditis with adhesions, myocardial changes were entirely absent in 13. Fifteen showed fatty degeneration, 14 fibrosis, and 7 brown atrophy. Careful study of these cases in particular was made in order that, if possible, some conclusions might be drawn as to the effect on the myocardium of pericardial adhesions. In so far as we can conclude in those instances in which extensive effects on the heart were associated with pericardial adhesions, the myocardial disease was in most cases clearly the earlier, and we fail to find in our series of cases what we have also failed to demonstrate clinically, namely, that adhesive pericarditis has any very important or serious effect on the anatomical condition of, or on the action of, the heart. In so far as we have been able to observe, some of the cases showing most pronounced adhesions clinically manifested little or no evidence of cardiac embarrassment *unless the myocardium itself was diseased,* usually primarily or independently so.

None of our six cases of true tuberculous pericarditis showed tuberculous invasion of the myocardium, and in none were the myocardial lesions apparently due to the pericarditis.

Renal changes are so universally present in all cadavers that it is very difficult to make an intelligible analysis of the occurrence of changes in these organs in association with pericarditis. Manifestly, in those cases showing arteriocapillary fibrosis alterations of this character were present in the kidneys, but in no instance were we able to find anything indicative of any inter-relationship between pericarditis and renal disease. Neither do we find from our study any indication that renal disease per se is an important factor in the causation of pericarditis, except in a very general or indirect way, for example, when, as suggested by Thorel, pericarditis arises as a terminal infection or perhaps follows myocardial disease of renal origin.

THE CAUSE OF DEATH. One of the most interesting parts of our study has been the investigation of the cause of death in pericarditis. This, we find, is only very rarely due to the pericarditis itself. In but 6 of our total of 150 cases was pericarditis mentioned as among the causes of death. In five of these instances the lesion was septic in nature, and in the one remaining, serofibrinous, due to a general infection, with its most pronounced focus in the pericardium. In most cases the causa mortis has been the general disease or condition in which the pericarditis had arisen as a complication. It is also interesting to note that notwithstanding the admitted frequency with which rheumatism acts as the cause of pericarditis, death was found to result from definitely rheumatic changes in but two instances, both examples of serofibrinous pericarditis.

Naturally, septic conditions are found to be the most frequent cause of death in pericarditis, and in 86 of our 150 cases death resulted from infections exclusive of tuberculosis and syphilis. In some of these, as in 17 cases of chronic adhesive pericarditis, the infections were apparently terminal in character; 8 of them were thus pneumonic and 9 simple sepsis. In the serofibrinous cases the infections were for the most part not only the cause of death, but also the obvious cause of the pericarditis. In the serofibrinous cases it is interesting to note that the cause of death was an inflammatory process adjacent to the pericardium in 30 instances of lobar pneumonia, 4 cases of bronchopneumonia, 1 case of empyema, and 9 cases of acute endocarditis. Not included in this list are four instances of death from acute myocarditis and four from acute tuberculosis.

In chronic adhesive pericarditis the cause of death appears to be absolutely independent of the pericardial lesion, which we have come to consider as of very little prognostic importance. Pericarditis was not even mentioned as among the causes of termination in any of these cases, and in but 5 instances was a cardiac dilatation the immediate cause of exitus. After a careful study of these cases, in three only can the pericardial inflammation or adhesions be considered as probably actively concerned in the cause of death. Chronic endocarditis was the terminal factor in 4 instances, acute endocarditis in 1, lobar pneumonia in 8, sepsis in 9, empyema in 1, and tuberculosis in 9. General changes attributable to alcoholism appear as the probable cause of death in 6 instances, and definitely syphilitic lesions in 4, myocarditis of non-syphilitic nature in 4, and traumatism in 2.

In the chronic fibrous form of pericarditis without adhesions the cause of death is even more indefinitely connected with the pericardial lesion, and, taken as a whole, we again wish to express our conviction of the very minor role which pericarditis in any form plays in prognosis, except as a measure of the degree or extent of any general infection.

CONCLUSIONS. In conclusion, we wish to summarize those points which have especially impressed themselves upon us in the course of this study, although some of the evidence leading us to these conclusions has been but incompletely expressed in the paper:

Pericarditis is a lesion secondary in nature, rarely or never primary. The complication, for such it must be considered, is in most cases not in itself serious except when of the suppurative variety, and in all instances it is more noteworthy as indicating the general condition in which it arises than on account of its own importance. It is, therefore, of little relative clinical importance, except as a diagnostic index, and in suppurative cases. True myocarditis is infrequently associated with pericarditis, but myocardial degeneration is commonly found, and is due in most instances not to the pericarditis, although generally caused by the same condition as the pericardial inflammation. Death rarely results in pericarditis from this lesion. Independent myocardial degeneration leading to dilatation of the heart, and especially fatty degeneration of the myocardium, is a predisposing or determining factor toward pericarditis. Overaction of the heart may induce pericardial inflammation. Serofibrinous pericarditis is in most instances an evidence of generalized bacteremia. Chronic adhesive pericarditis is a lesion of great frequency, often impossible of diagnosis, and in itself of very little clinical significance or importance. Serious symptoms arise from adhesive pericarditis only when the myocardium itself is seriously diseased, either concomitantly or quite independently. The signs usually cited as characteristic of pericardial synechia develop only when mediastinal inflammation or adhesions of marked degree are present in addition to the pericarditis.

NORMAL AUSCULTATORY DIFFERENCES BETWEEN THE SIDES OF THE CHEST.

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Differences at the Bases Behind. From those who are concerned to make the earliest possible diagnosis of pulmonary tuberculosis we hear more and more loudly each year the command to study with all possible care and attentiveness the auscultatory differences between corresponding portions of the two sides of the chest, as well as between different portions of the same lung. The slightest variation in pitch, quality, intensity, or duration of either inspiration or expiration at any point, as compared with conditions in the corresponding portion of the other lung, is, we are told, a sign to be noted, since it may be one of the factors in a decision affecting the patient's whole future life.

If this is true, and I see no good reason to doubt it, it behooves us to know as much as we can about any differences existing between the auscultatory phenomena in the right lung and those in the left in the healthy chest. It has been clearly recognized, at any rate since Austin Flint's time, and by the great majority of competent practitioners, that the apices of the two lungs differ in a large proportion of healthy persons, the apex of the right being more like that we expect to find in beginning solidification (from any cause) than is the apex of the left. All this is too familiar to need repetition. I wish, however, to call attention to what I believe to be a fairly common difference between the right and the left base posteriorly.

In a series of 250 healthy cases recently examined with reference to this point, I found the difference to which I allude to be present in 67 per cent. In these cases I noticed that the breathing at the left base was notably louder and harsher than that at the right. In the majority of these it had also the quality described by the Germans as "rough." I have been unable to see that age, sex, the time of day, or the amount of food in the stomach had any effect upon this phenomenon; yet I found it sometimes present in the morning and absent in the evening, for reasons unknown to me. It seems to be distinctly less common in persons under twenty years of age.

I have nothing of importance to say as to the cause of this difference. Thinking that it might be due to the amount of gas in the stomach, I inflated a patient's stomach with air and auscultated the backs of the lungs before and after the inflation, but I could not see that any considerable effect was produced by this procedure. One might conjecture that the slightly lower position of the diaphragm upon the right, or the greater impingement of